RECOVERY FROM TUBERCULAR MENINGITIS, WITH REPORT OF CASES.

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McCarthy in the *Phipps Institute Reports* for 1908 remarks "that evidences of recovery in tuberculosis of the nervous system is as rare as it is common in the lungs." That there is a retrograde process in tuberculosis of the glands, bones, peritoneum, and lungs is, of course, a trite observation.

In the nervous system occasionally tuberculous processes calcify, and meningeal infections become fibrous, quiescent, and finally heal. That such is the case in meningitis of tubercular origin has been a matter of comment by quite a number of writers, notably by Martin, in *Brain* for 1908, who has collected a series of cases in which recovery took place. In all text-books the prognosis is put down as grave or always grave. Dieulafoy mentions but one case that recovered, but he evidently had not scanned the literature thoroughly, because in his own country at the time at which his book was written there were records of at least six or seven cases of undoubted tubercular meningitis followed by recovery. But even the most optimistic must agree that it is always best to tell the family of one stricken with this disease that the outlook is practically hopeless; nevertheless, one must do so guardedly, and follow out certain lines of rational treatment, to be detailed later, in the hope that the lesions may be limited to the cortical membranes of the brain, without exudation of fibrin or purulent matter, and may not extend into the ventricles.

There are, of course, several meningeal diseases closely simulating tuberculosis, and these must be considered in several ways. These are, notably: (1) Quincke's disease, in which there is simply an excess of cerebrospinal fluid, chiefly in the ventricles, not produced by microörganisms and not infectious; (2) influenzal meningitis, simulating it closely, (3) and the meningismus of typhoid. Hereditary syphilis in the young, too, may resemble meningitis of tubercular origin. From the fact that Quincke's disease, which is not of parasitic origin, and therefore probably not toxic in the character of the serous exudate, can simulate true meningitis, it is evident that simple pressure within the ventricles and hence to the skull may cause nervous symptoms, chiefly cephalalgia in both disorders. In meningitis the added poisons and the multiple tubercles massed in nerve trunks and fibrous purulent exudate add much to the pathology and to the gravity of the prognosis.

A typical case of influenzal meningitis is the following: A young grocer, aged twenty-seven years, married, father of one healthy child, was one evening suddenly seized with a violent headache. This did not yield to any of the ordinary headache remedies, so that by morning morphine was required. Then it was seen from the retraction of the neck, intense pain, retracted belly, tâche cérébrale and Kernig's sign, fever, photophobia, and delirium, that meningitis was present. Accordingly, a lumbar puncture was done and 70 c.c. of clear liquid were drained off. This contained a fibrin clot, lymphatic cells exclusively, and no organisms. The headache immediately disappeared, Kernig's sign and rigidity of neck did so after ten days. He gained weight and resumed business. Two months afterward a second attack necessitated another puncture. He then had double vision on looking down and marked abduction of the eyes; jerking of the eyeball was demonstrable. Photophobia, increased knee-jerks, plantar flexion of the toes, tâche cérébrale, rigidity of the neck on the right side appeared. At this time headache did not seem so intense, and there was not so much fever. In removing 35 c.c. of fluid the double vision and fulness of the head disappeared during the operation, and the patient became markedly better. The fluid was limpid, clear, but contained some minute bacilli, probably influenzal. A guinea-pig injected with the fluid recovered. In twenty days all symptoms of any disorder promptly disappeared, save some stiffness of the neck. This was probably a case of influenzal meningitis, with lymphocytosis, cured by lumbar puncture and urotropin, which was freely given. There was no history of syphilis in the case, and the Wassermann test was negative.

The following is the history of a case of tubercular meningitis with recovery:

J. S., aged fifty years, linen draper, was admitted to St. Timothy's Hospital with every symptom of meningitis, from which he had suffered for four or five days. He had had such an intense headache that be begged to be shot, and indeed a revolver in his room had to be removed for fear that he would do so. When admitted he was delirious and had a retracted neck, scaphoid belly, and tâche cérébrale. A lumbar puncture was done under chloroform and 80 c.c. of fluid were withdrawn. He was a very tall man, measuring over six feet four inches, but was far from robust. In each apex there was evidence of tuberculosis, which from his history had existed for some time; in every way his history was that of one who had a low-grade phthisis for ten years. The present attack came on with violent vomiting and unendurable headache, also pains in his arms and legs. Kernig's, Oppenheim, and Babinski's signs were all present markedly. Pupils were contracted and rigid. On attempting to flex his neck after the lumbar puncture all the major joints flexed, and he groaned.

The lumbar puncture was easily performed, the fluid spurted out, and was quite bloody, due to the puncture of a small vein. This fluid contained three acid-fast bacilli, which in view of the presence of tuberculosis of the lungs were assumed to be tubercle bacilli. No animal injections were made. A cytodiagnosis was not attempted because of the admixture of blood. His pulse was 70; respiration, 26 and jerky; temperature, 99°. His leukocytes numbered 6200; red cells numbered 4,200,000; hemoglobin, 75 per cent. His urine was bloody, and contained albumin and casts. Blood pressure: 128 systolic; 88 diastolic. Marked improvement followed the withdrawal of the fluid. His spastic symptoms slowly receded. His mind became clear in a few days and his headache disappeared. He was given forced diet and urotropin, 30 grains a day. After three weeks' stay in the hospital, to the amazement of all, he suddenly got up, asked for his clothes, and went home. He then had some rigidity of his neck and some evidences of Kernig's phenomenon. After two years he is able to conduct his business and has no evidences whatsoever of any meningitis. In this instance the fact that the patient was an adult and long infected with a low-grade tuberculosis no doubt contributed much to his recovery.

Perhaps the most interesting case of recovery after tubercular meningitis is that reported by Rumple (quoted by Martin), who exhibited at the Aerztlichen Verein, Hamburg, the brain of a boy who died of phthisis. At the age of nine the boy had an acute attack of meningitis, in which there was retraction of the neck and hyperesthesia, absence of knee-jerks, bilateral optic neuritis, ptosis, and palsy of the external recti. Lumbar puncture gave a fluid in which tubercle bacilli were found. The boy, who was treated with repeated lumbar punctures, recovered, and though for some months was dull and stupid, he eventually retained his normal mental condition and was able to keep pace with other children of his age at school, while all physical signs had disappeared. Four years his health continued good, he then developed a tubercular abscess in the axilla, followed by phthisis; death took place eight years after the onset of the meningitis. At autopsy it was found that the membranes over the fissure of Sylvius on either side of the brain were thickened and glued together, but miliary tubercles were no longer visible. Not only was there a confirmation during life of the presence of tubercular meningitis, by the finding of tubercle bacilli, but also post mortem, at which the site of the old infection was discovered and definitely identified. Martin sums up the following requirements to be met in making a diagnosis of tubercular meningitis followed by recovery:

1. Clinical evidence of tuberculosis elsewhere in the body and history of exposure.
2. Differential count of cells in the spinal fluid (meaning an excess of lymphocytes).
3. Presence of tubercles in the choroid.
4. Tuberculin reaction.
5. Demonstration of bacilli in the fluid by staining, inoculation, and culture.

6. Postmortem examination after recovery or long remission and death by some other agency.

If tuberculosis of the meninges be but a local manifestation of a generalized infection (miliary tuberculosis) then the likelihood of recovery is of course minimized. That it is likely under such circumstances, is evidenced by cases reported by Lunn, (1) Dujardin-Beaumetz (2), Thornalas (1), and Brooks (2), in all of these tubercles were seen in the choroid, indicating a generalized infection. Tuberculin reaction, of course, confirmed the diagnosis, but it does not of necessity prove that there may not be some other remote latent infection which is the cause of the reaction. Postmortem examination and clinical diagnosis of tuberculosis elsewhere are, of course, convincing confirmation that the disease is tuberculosis.

In the case of a young woman, recently under the care of the writer, who had severe retractions of the neck, with pain, photophobia, increased knee-jerks, tâche cérébrale, tuberculous apex, bilateral pleurisy, high fever, and definite history of exposure to tuberculosis in her home, lumbar puncture revealed nothing and did not alleviate symptoms a particle. A later development of iritis and arthritis of the elbow compelled a revision of diagnosis to rheumatism of the spine, pleura, joints, and eye, showing that mere dependence upon the previous existence of a latent tuberculous lesion for confirmation was misleading and not dependable. Cytodiagnosis is also misleading. Porat reports cases showing that it may be valueless. Forbes, who examined 80 cases, 70 being verified by autopsy or by finding tubercle bacilli, found 51 had an excess of lymphocytes, 5 had an excess of polymorphonuclears; in 4 the proportion was equal. In tuberculosis of the brain not communicating with the meninges the fluid was normal. Goggia found an excess of lymphocytes in a meningococcus infection. Mutzner concludes, from a study of a series of cases with postmortems, that a lymphocytosis is not by any means diagnosed, and reports cases with an excess of the many nuclear forms of leukocytes.

In 797 cases collected by Martin for the London Hospital of unconfirmed tubercular meningitis, 16 (or 2 per cent.) recovered. In Vienna 1369 cases were reported in which recovery took place in 6 (or 4.4 per cent.). Thus in a total of 2166 cases, 22 (or about 1 per cent.) recovered. These, of course, were diagnosticated from mere clinical symptoms. Barlow, also quoted by Martin, states that if the tuberculous process be limited to a part of the surface of the brain there is a possibility of recovery, but if the disease, becomes generalized so that besides the invasion of the pia mater there is an extensive meningo-encephalitis, with or without hydrocephalus, the chances of recovery are practically nil.

McCarthy in several *Phipps Institute Reports* found frequent evidences of healed tuberculosis of the nervous system in subjects that had finally died of phthisis.

TRUE CASES.

Case.	Autopsy.	Bacilli found.	Tuberculosis elsewhere in body.	Cytodiagnosis.	Tubercles in choroid.	Clinical diagnosis.	Tuberculin.	Tuberculous history.	
Treyhan.	None, recovered	Yes	..	..	None	Yes	None	..	Choked disk.
Henkel, 17 yrs.	None, recovered	Yes	..	..	..	Yes	..		
Gross, 20 yrs.	None, recovered	Yes	Yes	Excess polys.	..	Yes	..	..	Developed phthisis 8 months after.
Stark, 44 yrs.	None	Yes	..	..	None	Yes	..	..	Optic neuritis.
Barth, 3 yrs.	None	Yes	None	None	None	..	..	..	Recovered in 6 months.
Stiles.	None	Yes	None	..	None	Yes	..	..	Recovered after decompression.
Stiles.	None	Yes	None	..	None	Yes	..	..	Recovered after decompression.
Alanzino.	None	Yes G. P.	None	Yes	..	Yes	..	..	Recovered in good health, 6 months.
Tedeschi, 14 yrs.	None	Yes	None	Yes	..	Yes			
Jemma, 3½ yrs.	None	Yes	None	Yes	..	Yes	..	..	After 3 years, good health.
Gareiso, 8 yrs.	None	Yes	None	Yes	None	Yes	..	..	Well 12 months after.
Claisse, 30 yrs. Abramc.	None	Yes	None	Yes	..	..	..	..	Satisfactory recovery.
Dufour.	Yes	..	..	..	..	Yes	..	..	Old tubercles found.
Dufour.	Yes	..	..	..	..	Yes	..	..	Old tubercles found.
Vaquez-Digne.	..	Yes G. P.	Yes	Yes	..	Yes			
Rossini.	..	Yes G. P.	..	..	..	Yes	..	..	Complete recovery.
Janssen, 19 yrs.	Yes	..	..	..	..	Yes	..	..	Meningitis 3 years before death from phthisis.
Politzer.	Yes	..	..	..	..	Yes	..	..	Old lesions and new lesions, 3-year interval between attacks.
Schwalbe.	Yes	..	..	..	..	Yes	..	..	Both died of diphtheria.
Schwalbe.	Yes	..	..	..	..	Yes	..	..	Evidence of old tubercles found at autopsy.
Carrington.	Yes	..	..	..	..	Yes	..	..	Died of psoas abscess.
Leube, 24 yrs.	Yes	..	..	..	..	Yes	..	..	Died of phthisis and recurrent meningitis. Old tubercular lesion of brain found from previous attack.
Riebold, 6 yrs.	None	G. P. Yes	..	..	..	Yes	..	..	574 c.c. drawn in punctures.
Rumpel, 17 yrs.	Yes	G. P. Yes	Yes	..	..	Yes	..	..	Died of phthisis 4 years after recovery from meningitis.
Lunn.	None	..	..	..	Yes	Yes	..	..	Meningitis followed by recovery.
Dujardin-Beaumetz.	None	None	None	..	Yes	Yes	..	..	Recovery; diagnosis based on choroid tubercle.
Thornallas.	None	None	Yes	None	Yes	Yes	..	..	Tubercles seen in choroid; disappeared on recov'y.
W. T. Brooks, 4 yrs.	Yes	Yes	Yes	Yes	Yes	Yes	Yes	..	Autopsy showed old healed meningitis; subsequently died after a long period.
Pitfield, 55 yrs.	None	Yes	Yes	Yes	None	Yes	None	Yes	Recovery, 3 weeks; old tuberculous lesion of the lungs.

I am able to append 29 cases of undoubted tuberculosis, mostly collected by Martin, in which recovery followed and 8 others more doubtful. In 10 autopsy confirmed the early diagnosis. In 18 tubercle bacilli were detected and in 5 of these guinea-pig inoculation confirmed the diagnosis. In 4 cases, tubercles of the choroid were seen. In 7 demonstrable tuberculosis elsewhere was noted.

In view of the fact that recovery can take place in perhaps 1 in 200 cases, steps should be undertaken actively to facilitate such a possibility. The patient should be put in a quiet, airy, dark place and kept as free from annoyance as possible. Lumbar puncture should be performed at once. The writer had a case, a boy, aged five years, from whose spine 40 c.c. of fluid were removed every other day for two weeks, with marked amelioration in the symptoms. Forced feeding through nasal tube, with milk and eggs, should be done. Morphine administered for pain and as a sedative, if respiration is not embarrassed thereby, and because free formaldehyde is found in the spinal fluid after the ingestion of urotropin the latter drug should be given freely. I have found, after giving it for one day, that formaldehyde can be detected in the fluid with iron and sulphuric acid; and because Raw has reported recovery in two cases in which tuberculin was used, I would advocate one or two injections of this remedy.

One case was most skilfully operated on by Dr. George Müller, who decompressed and punctured the left lateral ventricle, withdrawing about four times the normal amount of fluid, the child ultimately dying of respiratory palsy, having lived for three weeks after the diagnosis had been made and gaining five pounds by forced feeding. Why decompression is not fraught with better results in this disease is probably due to the involvement of the ventricles. The unrelieved distention is due to the choking of the canal leading from the third to the fourth ventricle.

AN INTENSIVE STUDY OF THE EPIDEMIOLOGY OF PELLAGRA. REPORT OF PROGRESS.[1]

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Part I.

I. General Plan of the Work. Although the Thompson-McFadden Pellagra Commission will continue its work in the field

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[1] From the Thompson-McFadden Pellagra Commission, New York Post-Graduate Medical School and Hospital.